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Anatomy of SUV

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ABSTRACT. Standardized uptake value (SUV) for [F-18]fluorodeoxyglucose (FDG) studies that is commonly used to differentiate malignant from benign tumors and to assess the efficacy of therapy is reviewed as a simplified calculation of the more general modeling approach. Based on such a basis, the merits and limitations of the SUV approach is examined with reference to literature reports on tumor uptake of FDG. Results indicate the complexity and large variation of glucose uptake mechanism in tumors. Consistently performed procedures and more basic studies are needed to improve the utility of FDG SUV. NUCL MED BIOL 27;7:643–646, 2000. **© 2000 Elsevier Science Inc. All rights reserved.**

KEY WORDS. Tumor, Glucose utilization rate, Kinetic modeling, Tracer

INTRODUCTION

Standardized uptake value (SUV), under various names, is a popular index used in clinical [F-18]fluorodeoxyglucose (FDG) studies to differentiate malignant from benign tumors (1, 2, 10, 11, 15, 19, 21, 23, 25, 26, 31) and to assess the efficacy of therapy (5, 9, 28). Despite its popularity, the reliability of SUV is still somewhat controversial (17). In the following, I will compare the SUV method with modeling approach to examine its merits as well as its variability. Issues related to physical measurements, such as attenuation correction, object size, imaging resolution, and regions of interest (ROI), that have been addressed separately by others will not be discussed extensively here.

Common factors that can influence the amount of tracer uptake in tissue are listed in Table 1. In addition to the primary biochemical process, there are many other confounding factors that can influence the amount of tracer uptake. Although not every factor listed in Table 1 would play a role for every tracer, they all potentially could affect significantly the tissue uptake of a particular tracer. Tracer kinetic modeling that describes mathematically the mechanism of transport and biochemical reactions of the tracer in tissue is a formal way to remove the effects of the confounding factors (13). Modeling approach usually requires taking series of blood samples from the studied subject to give the time course of the tracer delivery, and requires measuring the dynamics of the radiolabel in local tissues. Frequently, model fitting or regression analysis is needed to give the desired biological information.

For some tracers and for some studies, there are simplified approaches that can achieve a similar goal of reducing the effect of confounding factors. The SUV for FDG can be viewed as one of these approaches. Although the SUV formula in its original form has been criticized (17), the simplicity of the approach makes it extremely attractive for routine clinical use. Changes/modifications of the formula have been made to overcome the original deficiencies (8, 17, 18, 36). The following is a review of the basis of the approach to show how it is related to the modeling approach. Based on such a foundation, the merits and limitations of the SUV approach (with its various modifications) can be assessed more easily.

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RELATIONSHIP BETWEEN GLUCOSE UTILIZATION RATE AND SUV

Quantitation of glucose utilization rate with FDG (14, 27) followed the original work of Sokoloff *et al.* (30) who had laid out a solid foundation for the use of C-14-labeled deoxyglucose (DG) and autoradiograph for the quantitation of cerebral metabolic rate of glucose. In this method, the time course of tracer delivery to local tissue is provided by plasma concentration of FDG. The transport of FDG across the capillary/cellular membrane is accounted for by two rate constants K_1 and k_2 (for forward and reversed transport). The phosphorylation of FDG to FDG-6-P in cells is accounted for by a rate constant k_3, and a usually small dephosphorylation rate constant for FDG-6-P is represented by k_4. With this representation, the glucose utilization rate in tissue can be formulated as seen in Eq. (1):

$$MRglc = ([Glc]/LC) \cdot [K_1 \cdot k_3/(k_2 + k_3)] = ([Glc]/LC) \cdot K_i \quad (1)$$

where LC is a lumped constant that accounts for the transport and phosphorylation difference between FDG and glucose (12, 14, 27, 30), and [Glc] is the glucose concentration in arterial plasma. K_i is defined as $[K_1 \cdot k_3/(k_2 + k_3)]$ and is commonly called the uptake constant. Once the value of K_i is known, MRglc can be easily obtained. There are numerous ways to estimate K_i (13, 24, 30), but the one provided by Sokoloff *et al.* (30) is most revealing, and can be described by the following descriptive formula (14, 30) [see Eq. (2)]:

$$K_i = [(\text{total radioactivity concentration in tissue at time } T) - (\text{free FDG in tissue at time } T)]/[\text{integral (up to time } T) \text{ of the time course of available FDG concentration for tissue uptake}] \quad (2)$$

Normally, when T is reasonably large (*e.g.*, larger than 45 min postinjection), the tissue's free FDG is a small fraction of the total radioactivity in tissue and can be neglected. The time integral in

Table 1. Common Factors That Affect Tracer Uptake in Tissues

Amount of dose administered
Route of tracer administration
Specific biochemical processes that the tracer probes
Blood perfusion of the tissue of interest
Body size of the subject
Systemic condition of the body (uptake of tracer in other tissue/organs, excretion rate of the tracer, body fat content, biochemical reaction/metabolism of the tracer in the body, etc.)
Endogenous substrate/transmitter competition
Nonspecific biochemical environment in tissue
Biochemical properties of labeled metabolites in plasma
Vascular volume in tissue
Time of uptake determination after tracer administration

the denominator can also be approximated as the integral of the time activity curve (TAC) of FDG concentration in arterial plasma. Therefore, the equation for MRglc is reduced to [see Eq. (3)]:

$$\text{MRglc} = [\text{Glc}] \cdot C(T) \Big/ \left\{ \text{LC} \cdot \left[\int_0^T (\text{plasma FDG TAC})\, dt \right] \right\} \tag{3}$$

where $C(T)$ denotes the radioactivity concentration in tissue at time T that is measurable externally with PET or coincidence camera, and its spatial distribution in tissue is what an FDG PET image normally represents.

If the time integral in the denominator is proportional to the injected dose divided by body weight of the subject, then [see Eq. (4)]:

$$\left[\int_0^T (\text{plasma FDG TAC})\, dt \right] = b \cdot (\text{dose})/(\text{body weight}) \tag{4}$$

with b as a proportional constant that is not dependent on the particular subject being studied. Therefore, the equation for MRglc becomes [see Eq. (5)]:

$$\text{MRglc} = \{([\text{Glc}]/100) \cdot C(T)/(\text{dose/body weight})\}/(\text{LC} \cdot b/100) \tag{5}$$

The quantity within the braces is SUV of the glucose-corrected form (with mg/dL as the units of [Glc]). Since LC is a constant (14, 27, 30) and b is also assumed to be constant, SUV should thus be proportional to MRglc and thus can be used as an index for MRglc. This provides a theoretical foundation of SUV. A good correlation between SUV and MRglc has been shown for many types of tumors, including sarcoma (8), bronchial carcinoma (19), and head and neck tumors (20).

Some SUV formulae that do not include the [Glc] factor cannot account for variation of plasma glucose variation from subject to subject, as pointed out by Keyes (17), although there are situations for which the formulae without the [Glc] factor might be more appropriate (see later discussion). The approximation of the integral of plasma FDG TAC to be inversely proportional to body weight has also been questioned, and the use of "lean body weight" (36) or "body surface area" (18, 29) to replace "body weight" has been offered and is now also frequently used.

VARIABILITY OF SUV

As shown in the above derivation of the SUV formula, the validity of SUV depends on a few important assumptions and approximations. The most critical one is the validity of the approximation of the time integral of plasma FDG TAC with Eq. (4). It does not require the plasma FDG TAC to have a fixed shape, but the integral of the curve is assumed to be proportional to the injected dose and inversely proportional to body weight or body surface area. While these assumptions are quite reasonable, the equation implies that the integral of plasma FDG TAC is not affected significantly by other factors that are variable from subject to subject. Ishizu *et al.* (16) examined this issue for patients with glucose-loading hyperglycemia. Based on their published data in 10 patients, the ratio of the time integral and (dose/body weight) is 1.84 ± 0.36 (*i.e.*, coefficient of variance is below 20%). The SUV calculation (without including plasma [Glc]) has a good correlation with the FDG uptake value that is normalized by the time integral of the plasma TAC (correlation coef = 0.82). So, in general, this is a reasonable assumption for FDG. However, for instance, if a patient is going through chemotherapy and has impaired renal function, the clearance of plasma FDG through the kidney could be significantly reduced, and the integral of the plasma TAC could deviate more from what would be predicted from the dose and body weight alone. The SUV value for such a case could overestimate the tumor MRglc, and the therapy response may not be accurately reflected by the change in SUV.

Conceptually, it appears that such a change in the FDG TAC would also affect the amount of tracer uptake in other background tissues. In other words, the tumor to background ratio on the FDG images might not be affected by the plasma FDG TAC changes, and the tumor to background ratio might be used as an adjunct to monitor the reliability of SUV. The validity of this argument of course depends on the uptake kinetics of FDG in the background region. Unfortunately, the FDG kinetics in the background could differ markedly from those in tumors. For example, the FDG kinetics in normal liver tissue have such a large dephosphorylation rate (k_4) that the radioactivity in tissue at late time is related more to the plasma FDG concentration at that time rather than to the integral of the FDG TAC (3, 22). Also, the FDG uptake characteristics and glucose utilization in many tissues could be affected differently by other factors, such as plasma glucose (4) and body weight (36). In an animal study to examine the effect of hyperglycemia, Wahl *et al.* (34) reported that as plasma glucose is increased, the FDG uptake in brain, small bowel, and ovaries are decreased, while the uptake in kidney is increased. Lindholm *et al.* (20) also found that tongue and muscle uptakes are increased with plasma glucose. Under insulin-induced hypoglycemia in experimental animals, Torizuka *et al.* (33) showed that changes in FDG uptake varied greatly in different tissues. Thus, the FDG uptake in background regions is usually not stable enough to serve as a reliable reference. The tumor to background ratio has been examined for its ability to differentiate malignant from benign tumors and was found to be less reliable than SUV (4, 6, 7).

A question that is frequently raised about the use of FDG for measurement of MRglc is the stability of LC. One may wonder how that would affect the SUV. LC represents the relative overall uptake efficiency between FDG and glucose. Since FDG uptake in tissue usually goes through two limiting steps and the relative

efficiency between FDG and glucose are different, the relative overall uptake efficiency between FDG and glucose that is represented by LC could change when the dominance of the two limiting steps is changed. For example, in normal brain tissue, FDG has a higher rate than glucose for the transport across the blood-brain barrier, while the reverse is true for the phosphorylation step. Also, since cerebral MRglc is regulated to remain at a rather constant rate, the dominance between the two limiting steps could be shifted when plasma glucose concentration is shifted between its extremes. Thus, the overall relative uptake rate would be dependent on which step is more limiting. However, within the normal physiological range of plasma glucose concentration, the value of LC in brain tissue does not vary much. If the FDG uptake in a particular tissue has only one single limiting step, LC is not expected to change at all. Therefore, potential variability of LC is not a major concern for normal use of the SUV.

As mentioned earlier, the SUV formula used by some people does not include the [Glc]/100 factor in Eq. (5). This has been a point of some controversy (17), and is actually related to the fundamental question of whether MRglc is the right index to characterize the activity of a tumor. For example, if MRglc in tissue is proportional to the plasma glucose level, as in skeletal muscle, the measured MRglc would vary depending on the plasma glucose level, and the SUV formula without the [Glc] factor would actually give a more stable parameter that is independent of plasma glucose variations. On the other hand, if the glucose transport/metabolic process in tissue is highly saturated or regulated, MRglc would remain constant, while the uptake constant K_i is inversely related to [Glc]. So, in this case, the SUV formula would need to include the [Glc] factor to compensate for the inverse relationship between K_i and [Glc]. Therefore, it is important to understand the glucose utilization characteristics of the particular tumor to select the appropriate formula to use. Work on a few common types of tumor has been performed and reported in the literature. For example, Diederichs *et al.* (7) showed the uptake of FDG in pancreatic tumors decreased at increased plasma glucose level. For glioma and bronchial carcinomas, similar plasma glucose dependencies have been shown (16, 19). So, for most tumors, the SUV formula that includes the [Glc] factor appears to be more appropriate. However, animal studies performed by Torizuka *et al.* (33) indicated that for insulin-induced hypoglycemia, the uptake of FDG in mammary carcinoma is also reduced, and thus including the [Glc] factor in this case would make the SUV further suppressed. Yamada *et al.* (35) showed that FDG uptake in transplanted ascitic hepatoma remained unchanged with plasma glucose variations. Torizuka *et al.* (32) also showed that in breast cancer, the SUV without plasma glucose adjustment correlated with both K_i and k_3 (phosphorylation rate constant), but in lung cancers SUV correlated only with K_i (*i.e.*, not with k_3). These results illustrate the complexity of glucose uptake in tumors, and more basic studies are needed to increase our understanding and to improve the utility of FDG SUV for differentiating malignant from benign tumors.

Physical imaging factors could also contribute to large variability in the calculated SUV. A major factor is the partial volume effect that is due to the variable tumor size relative to spatial resolution of the imaging device. The selection of ROI and alignment of images between follow-up and pretreatment studies could directly affect the SUV calculations. Attenuation correction is another important factor since accurate attenuation correction is needed to give reliable tissue radioactivity concentration. Also, since the calculation of SUV involves quantities measured with two instruments (tissue FDG uptake by positron emission tomography or coincidence camera and injected dose by radiodosimeter), calibration inaccuracy between the two instruments could contribute to variability of the SUV calculation, especially for comparison across different institutions/centers. In addition, radioactivity concentration in tissue during an FDG study does not remain constant even after 45 min postinjection and after correction for radioactive decay, because the continuing tissue uptake of FDG from plasma and the clearance of tissue activity from tissue are rarely in balance. Meanwhile, the entire body of the patient cannot be imaged at the same time by scanners available today. Therefore, variation in the time of the tissue uptake measurement used in the SUV calculation could introduce additional variability (17). Therefore, in addition to the consideration of the biological factors and tracer kinetic issues, the FDG study procedure and image analysis also need to be consistently and carefully performed to improve the clinical utility of SUV.

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References

1. Adler L. P., Blair H. F., Makley J. T., Williams R. P., Joyce M. J., Leisure G., al-Kaisi N. and Miraldi F. (1991) Noninvasive grading of musculoskeletal tumors using PET. *J. Nucl. Med.* **32,** 1508–1512.
2. Adler L. P., Blair H. F., Williams R. P., Pathria M. N., Makley J. T., Joyce M. J., al-Kaisi N. and Miraldi F. (1990) Grading liposarcomas with PET using [F-18]FDG. *J. Comput Assist Tomogr.* **14,** 960–962.
3. Choi Y., Hawkins R. A., Huang S. C., Brunken R. C., Hoh C. K., Messa C., Nitzsche E. U., Phelps M. E. and Schelbert H. R. (1994) Evaluation of the effect of glucose ingestion and kinetic model configurations of FDG in the normal liver. *J. Nucl. Med.* **35,** 818–823.
4. Crippa F., Gavazzi C., Bozzetti F., Chiesa C., Pascali C., Bogni A., De Sanctis V., Decise D., Schiavini M., Cucchetti G. and Bombardieri E. (1997) The influence of blood glucose levels on [F-18]fluorodeoxyglucose (FDG) uptake in cancer: A PET study in liver metastases from colorectal carcinomas. *Tumori* **83,** 748–752.
5. Dehdashti F., Flanagan F., Mortimer J. E., Katzenellenbogen J. A., Welch M. J. and Siegel B. A. (1999) Positron emission tomographic assessment of metabolic flare to predict response of metastatic breast cancer to antiestrogen therapy. *Eur. J. Nucl. Med.* **26,** 51–56.
6. Delbeke D., Martin W. H., Sandler M. P., Chapman W. C., Wright J. K. and Pinson C. W. (1998) Evaluation of benign vs malignant hepatic lesions with positron emission tomography. *Archives of Surgery* **133,** 510–515.
7. Diederichs C. G., Staib L., Glatting G., Beger H. G. and Reske S. N. (1998) FDG PET: Elevated plasma glucose reduces both uptake and detection rate of pancreatic malignancies. *J. Nucl. Med.* **39,** 1030–1033.
8. Eary J. F. and Mankoff D. A. (1998) Tumor metabolic rates in sarcoma using FDG PET. *J. Nucl. Med.* **39,** 250–254.
9. Haberkorn U., Bellemann M. E., Altmann A., Gerlach L., Morr I., Oberdorfer F., Brix G., Doll J., Blatter J. and van Kaick G. (1997) PET 2-gluoro-2-deoxyglucose uptake in rat prostate adenocarcinoma during chemotherapy with gemcitabine. *J. Nucl. Med.* **38,** 1215–1221.
10. Hamberg L. M., Hunter G. T., Alpert N. M., Choi N. C., Babich J. W. and Fischman A. J. (1994) The dose uptake ratio as an index of glucose metabolism: Useful parameter or over simplification. *J. Nucl. Med.* **35,** 1308–1312.
11. Hawkins R. A., Choi Y., Huang S. C., Messa C., Hoh C. K. and Phelps M. E. (1992) Quantitating tumor glucose metabolism with FDG and PET. *J. Nucl. Med.* **33,** 339–344.
12. Hawkins R. A., Phelps M. E. and Huang S. C. (1986) Effects of temporal sampling, glucose metabolic rates and disruptions of the blood brain barrier on the FDG model with and without a vascular compartment: Studies in human brain tumors with PET. *J. Cereb. Blood Flow Metabol.* **6,** 170–183.
13. Huang S. C. and Phelps M. E. (1985) Principles of tracer kinetic modeling in positron emision tomography and autoradiography. In: *Positron Emission Tomography and Autoradiography* (Edited by Phelps

M. E., Mazziotta J. and Schelbert H. R.) pp. 287–346. Raven Press, New York.
14. Huang S. C., Phelps M. E., Hoffman E. J., Sideris K., Selin C. J. and Kuhl D. E. (1980) Noninvasive determination of local cerebral metabolic rate of glucose in man. *Am. J. Physiol.* **238,** E69–E82.
15. Inokuma T., Tamaki N., Torizuka T., Magata Y., Fujii M., Yonekura Y., Kajiyama T., Ohshio G., Imamura M. and Konishi J. (1995) Evaluation of pancreatic tumors with positron emission tomography and F-18 fluorodeoxyglucose: Comparison with CT and US. *Radiology* **195,** 345–352.
16. Ishizu K., Nishizawa S., Yonekura Y., Sadato N., Magata Y., Tamaki N., Tsuchida T., Okazawa H., Miyatake S., Ishikawa M., Kikuchi H. and Konishi J. (1994) Effects of hyperglycemia on FDG uptake in human brain and glioma. *J. Nucl. Med.* **35,** 1104–1109.
17. Keyes J. W. (1995) SUV: Standard uptake or silly useless value. *J. Nucl. Med.* **36,** 1836–1839.
18. Kim C. K., Gupta N. C., Chandramouli B. and Alavi A. (1994) Standardized uptake values of FDG: Body surface area correction is preferable to body weight correction. *J. Nucl. Med.* **35,** 164–167.
19. Langen K.-J., Braun U., Kops E. R., Herzog H., Kuwert T., Nebeling B. and Feinendegen L. E. (1993) The influence of plasma glucose levels on fluorine-18-fluroroglucose uptake in bronchial carcinomas. *J. Nucl. Med.* **34,** 355–359.
20. Lindholm P., Minn H., Leskinen-Kallio S., Bergman J., Ruotsalainen U. and Joensuu H. (1993) Influence of the blood glucose concentration on FDG uptake in cancer—A PET study. *J. Nucl. Med.* **34,** 1–6.
21. Lowe V. J., Hoffman J. M., DeLong D. M., Patz E. F. and Coleman R. E. (1994) Semiquantitative and visual analysis of FDG-PET images in pulmonary abnormalities. *J. Nucl. Med.* **35,** 1771–1776.
22. Messa C., Choi Y., Hoh C. K., Jacobs E. L., Glaspy J. A., Rege S., Nitzsche E., Huang S. C., Phelps M. E. and Hawkins R. A. (1992) Quantification of glucose utilization in liver metastases: Parametric imaging of FDG uptake with PET. *J. Comput. Assist. Tomogr.* **16,** 684–689.
23. Minn H., Joensuu H., Ahonen A. and Klein P. (1988) Fluorodeoxyglucose imaging: A method to assess the proliferative activity of human cancer *in vivo*: Comparison with DNA flow cytometry in head and neck tumors. *Cancer* **61,** 1776–1781.
24. Patlak C. S., Blasberg R. G. and Fenstermacher J. D. (1983) Graphical evaluation of blood-to-brain transfer constants from multiple-time uptake data. *J. Cereb. Blood Flow Metab.* **3,** 1–7.
25. Patz E. F. Jr, Lowe V. J., Hoffman J. M., Paine S. S., Harris L. K. and Goodman P. C. (1994) Persistent or recurrent bronchogenic carcinoma: Detection with PET and 2-[F-18]-2-deoxy-D-glucose. *Radiology* **191,** 379–382.
26. Patz E. F. Jr, Lowe V. J., Hoffman J. M., Paine S. S., Burrowes P., Coleman R. E. and Goodman P. C. (1993) Focal pulmonary abnormalities: Evaluation with F-18 fluorodeoxyglucose PET scanning. *Radiology* **188,** 487–490.
27. Phelps M. E., Huang S. C., Hoffman E. J., Selin C. E. and Kuhl D. E. (1979) Tomographic measurement of regional cerebral glucose metabolic rate in man with (F-18) fluorodeoxyglucose: Validation of method. *Ann. Neurol.* **6,** 371–388.
28. Romer W., Hanauske A. R., Ziegler S., Thodtmann R., Weber W., Fuchs C., Enne W., Herz M., Nerl C., Garbrecht M. and Schwaiger M. (1998) Positron emission tomography in non-Hodgkin's lymphoma: Assessment of chemotherapy with fluorodeoxyglucose. *Blood* **91,** 4464–4471.
29. Sadato N., Tsuchida T., Nakaumra S., Waki A., Uematsu H., Takahasi N., Hayashi N., Yonekura Y. and Ishii Y. (1998) Non-invasive estimation of the net influx constant using the standardized uptake value for quantification of FDG uptake of tumours. *Eur. J. Nucl. Med.* **25,** 559–564.
30. Sokoloff L., Reivich M., Kennedy C., Des Rosiers M. H., Patlak C. S., Pettigrew K. D., Sakurada O. and Shinohara M. (1977) The [C-14]deoxyglucose method for the measurement of local cerebral glucose utilization: Theory, procedure, and normal values in the conscious and anesthetized albino rat. *J. Neuro Chem.* **28,** 897–916.
31. Strauss L. G., Clorius J. H., Sclag P., Lehner B., Kimmig B., Engenhart R., Marin-Grez M., Helus F., Oberdorfer F., Schmidlin P. and Van Kaick G. (1989) Recurrence of colorectal tumors: PET evaluation. *Radiology* **170,** 329–332.
32. Torizuka T., Zasadny K. R., Recker B. and Wahl R. L. (1998) Untreated primary lung and breast cancers: Correlation between F-18 FDG kinetic rate constants and findings of in vitro studies. *Radiology* **207,** 767–774.
33. Torizuka T., Fisher S. J. and Wahl R. L. (1997) Insulin-induced hypoglycemia decreases uptake of 2-[F-18]fluoro-2-deoxy-D-glucose into experimental mammary carcinoma. *Radiology* **203,** 169–172.
34. Wahl R. L., Henry C. A. and Ethier S. P. (1992) Serum glucose: Effects on tumor and normal tissue accumulation of 2-[F-18]fluoro-2-deoxy-D-glucose in rodents with mammary carcinoma. *Radiology* **183,** 643–647.
35. Yamada K., Endo S., Fukuda H., Abe Y., Yoshioka S., Itoh M., Kubota K., Hatazawa J., Satoh T., Matsuzawa T., Ido T., Iwata R., Ishiwata K. and Takahashi T. (1985) Experimental studies on myocardial glucose metabolism of rats with 18F-2-fluoro-2-deoxy-D-glucose. *Eur. J. Nucl. Med.* **10,** 341–345.
36. Zasadny K. R. and Wahl R. L. (1993) Standardized uptake values of normal tissues at PET with 2-[fluorine-18]-fluoro-2-deoxy-D-glucose: Variations with body weight and a method for correction. *Radiology* **189,** 847–850.